A Dissertation on

ANTI- HELICOBACTER PYLORI THERAPY RESPONSE IN EROSIVE AND NON EROSIVE GASTRITIS

Dissertation Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI – 600 032

With partial fulfillment of the regulations

for the award of the degree of

M.D. GENERAL MEDICINE

BRANCH – I

COIMBATORE MEDICAL COLLEGE
COIMBATORE
APRIL 2016

DECLARATION

I solemnly declare that this dissertation entitled **"ANTI-HELICOBACTER PYLORI THERAPY RESPONSE IN EROSIVE AND NON EROSIVE GASTRITIS"** was done by me at **Coimbatore Medical College and Hospital** during the **academic year 2013-2016** under the guidance and supervision of **Prof. Dr. M. RAVEENDRAN M. D.** This dissertation is submitted to The Tamil Nadu Dr. M. G. R. Medical University, towards the fulfillment of requirement for the award of M.D. Degree in General Medicine (Branch -I)

Place: Coimbatore

Date:

Dr. AHAMED SUBIR H

ACKNOWLEDGEMENT

At the outset, I would like to express my sincere gratitude to our Dean, **Prof. Dr. EDWIN JOE**, **M.D**., for his permission to conduct this study in Coimbatore Medical College.

I'm immensely grateful and indebted to my **Prof. Dr. KUMAR NATARAJAN M.D.,** Professor and Head, Department of Medicine, Coimbatore Medical College.

It gives me immense pleasure to express my sincere and deep gratitude to my guide, **Prof. Dr. M. RAVEENDRAN M.D.,** Professor, Department of General Medicine, Coimbatore Medical College, for his guidance and constant encouragement throughout the study.

It is with deepest sense of gratitude and respect that I would like to thank my co- guide, **Prof. Dr. S. RAJA M.D., D.M.,** Assistant Professor, Department of Gastro enterology, Coimbatore Medical College, for his constant support, guidance, invaluable suggestions and help that he has rendered through out the study.

I would like to acknowledge with gratitude, the help provided by our professors **Dr Usha M.D. and Dr. Sivakumar M.D.,** our unit Assistant Professors - **Dr. Geetha M.D**, **Dr. Sivakumar M.D.** and **Dr Akila M.D**. I would like to thank them for their valuable suggestions throughout the study. I'm indebted to them for being a constant source of inspiration.

I'm gratefully indebted to **Dr. Ravi Shankar M.D., D.M.,** Head and Professor, Department of Gastroenterology, for permitting me for conducting the

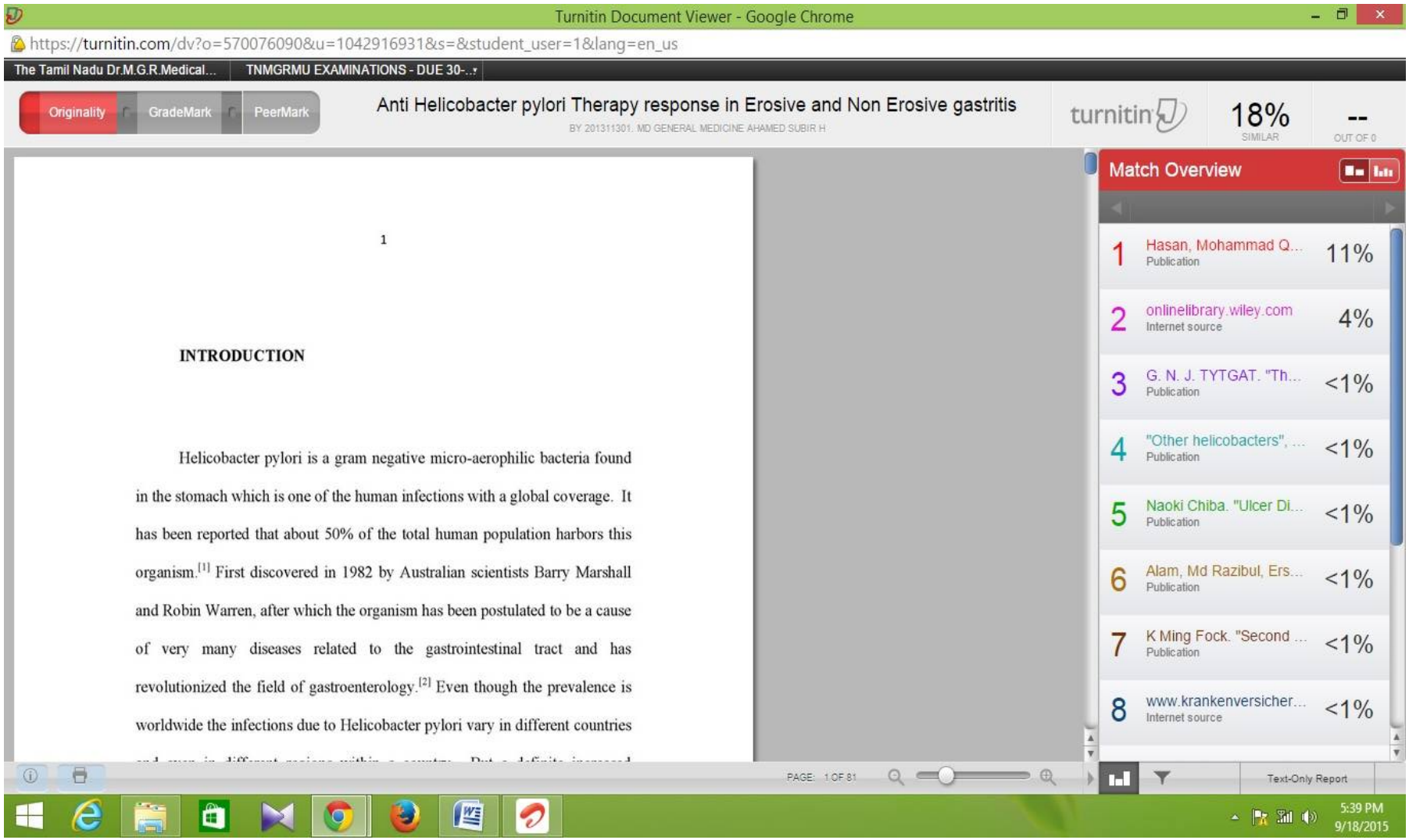
study and helping me with the study results, without which I would not have completed my study.

I would also like to thank **Mr. Jayakumar,** Lab assistant cum statistician , Department of Biochemistry,Trivandrum Medical College, Trivandrum ,Kerala in helping me with the statistical analysis for the study. I would always like to remember with extreme sense of thankfulness for the cooperation and criticism from my fellow post- graduates, my dear seniors and juniors .

I would like to thank my wife, **Dr. Rashma Mohammed P** for her constant support. I would like to take this opportunity to show my gratitude to my father, **Dr Humayoon Kabir S,** my mother Dr**. M. Saboora Beegum,** my father in law **Dr. P. A Mohammed Kunju**, my mother in law **Mrs Shameema** and my brother **Mohammed Samir H** and daughter **Imana Ahamed** for their never ending support and prayers.

I'm ever grateful to the **ALMIGHTY GOD** for showering his blessings on me and my family and I pray Almighty God to give me the strength to achieve all my endeavors.

Finally, I whole-heartedly thank all **my patients**, who formed the backbone of the study without which this would not have become a reality.

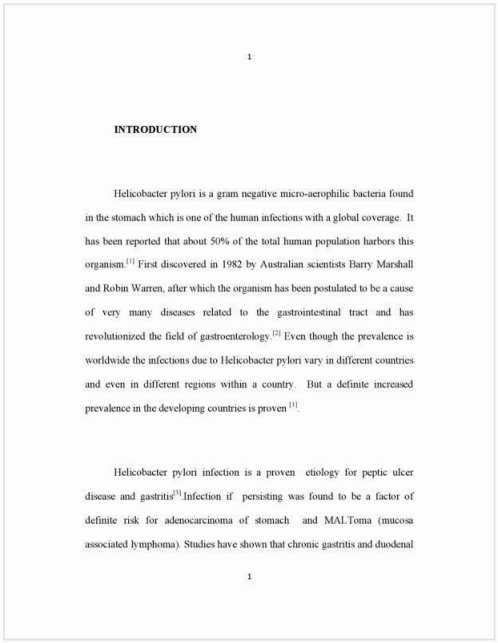

Turnitin Document Viewer - Google Chrome
https://turnitin.com/dv?o=570076090&u=1042916931&s=&student_user=1&lang=en_us
The Tamil Nadu Dr.M.G.R.Medical...
TNMGRMU EXAMINATIONS - DUE 30-...
Originality
GradeMark
PeerMark
Anti Helicobacter pylori Therapy response in Erosive and Non Erosive gastritis
BY 201311301, MD GENERAL MEDICINE AHAMED SUBIR H
turnitin
18%
SIMILAR
--
OUT OF 0
1
INTRODUCTION
Helicobacter pylori is a gram negative micro-aerophilic bacteria found in the stomach which is one of the human infections with a global coverage. It has been reported that about 50% of the total human population harbors this organism.[1] First discovered in 1982 by Australian scientists Barry Marshall and Robin Warren, after which the organism has been postulated to be a cause of very many diseases related to the gastrointestinal tract and has revolutionized the field of gastroenterology.[2] Even though the prevalence is worldwide the infections due to Helicobacter pylori vary in different countries
Match Overview
1 Hasan, Mohammad Q... Publication 11%
2 onlinelibrary.wiley.com Internet source 4%
3 G. N. J. TYTGAT. "Th... Publication <1%
4 "Other helicobacters", ... Publication <1%
5 Naoki Chiba. "Ulcer Di... Publication <1%
6 Alam, Md Razibul, Ers... Publication <1%
7 K Ming Fock. "Second ... Publication <1%
8 www.krankenversicher... Internet source <1%
PAGE: 1 OF 81
Text-Only Report
5:39 PM
9/18/2015

Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	201311301. Md General Medicine A…
Assignment title:	TNMGRMU EXAMINATIONS
Submission title:	Anti Helicobacter pylori Therapy re …
File name:	REVIEW_OF_LITERATURE_THESI…
File size:	1.11M
Page count:	81
Word count:	7,403
Character count:	40,831
Submission date:	18-Sep-2015 05:30PM
Submission ID:	570076090



1

INTRODUCTION

Helicobacter pylori is a gram negative micro-aerophilic bacteria found in the stomach which is one of the human infections with a global coverage. It has been reported that about 50% of the total human population harbors this organism.[1] First discovered in 1982 by Australian scientists Barry Marshall and Robin Warren, after which the organism has been postulated to be a cause of very many diseases related to the gastrointestinal tract and has revolutionized the field of gastroenterology.[2] Even though the prevalence is worldwide the infections due to Helicobacter pylori vary in different countries and even in different regions within a country. But a definite increased prevalence in the developing countries is proven [1].

Helicobacter pylori infection is a proven etiology for peptic ulcer disease and gastritis[3] Infection if persisting was found to be a factor of definite risk for adenocarcinoma of stomach and MALToma (mucosa associated lymphoma). Studies have shown that chronic gastritis and duodenal

1

ABBREVIATIONS

GIT	-	Gastro intestinal Tract
OGD	-	Oesophago gastro duodenoscopy
APCGH	–	Asia Pacific Consensus Guidelines
APD	–	Acid Peptic Disease
RUT	–	Rapid Urease Test
UBT	-	Urease Breath test
OLGA	-	Operative Link for Gastritis assessment
PPI	-	Proton Pump Inhibitors
H2 blocker	-	Type 2 Histamine receptor blockers
ITP	-	Idiopathic thrombocytopenic pupura
GERD	–	Gastro Esophageal Reflux disease
RF	–	Rheumatoid factor
ESR	–	Erthrocyte sedimentation rate
CRP	–	C reactive protein
HBV	–	Hepatitis B virus
HCV	–	Hepatitis C virus
HIV	–	Human immunodeficiency virus
NSAIDS	–	Non steroidal anti- inflammatory drugs
SLE	–	Systemic lupus erythematosus

WBC	–	White blood cell
CBC	–	Complete blood count
LFT	–	Liver function tests
RFT	–	Renal function tests
DNA	–	Deoxy ribonucleic acid
OPD	–	Out patient department
CLO	-	Campylobacter – like organism
MALT	-	Mucosa Associated lymphoid Tissue
Hcp	-	Helicobacter cysteine rich proteins
Vac A	-	Vacoulating cytotoxin A
Cag A	-	Cytotoxin associated gene A
SES	-	Socioeconomic Status
ITT	-	Intention-to-treat

LIST OF TABLES

LIST OF FIGURES

CONTENTS

INTRODUCTION

Helicobacter pylori is a gram negative micro-aerophilic bacteria found in the stomach which is one of the human infections with a global coverage. It has been reported that about 50% of the total human population harbors this organism.[1] First discovered in 1982 by Australian scientists Barry Marshall and Robin Warren, after which the organism has been postulated to be a cause of very many diseases related to the gastrointestinal tract and has revolutionized the field of gastroenterology.[2] Even though the prevalence is worldwide the infections due to Helicobacter pylori vary in different countries and even in different regions within a country. But a definite increased prevalence in the developing countries is proven [1].

Helicobacter pylori infection is a proven etiology for peptic ulcer disease and gastritis[3].Infection if persisting was found to be a factor of definite risk for adenocarcinoma of stomach and MALToma (mucosa associated lymphoma). Studies have shown that chronic gastritis and duodenal ulcer has an association of 100% compared to 50% in the controls who did not have ulcer [4]. Increased association of Helicobacter pylori was dependent on the type of gastritis and also depended on site of infection as shown by a prevalence of 57% in erosive gastritis, 28.7% in superficial gastritis, 63% in gastric erosion , 52.4% in case of early gastric carcinoma

and 80% in case of gastric ulcer [5]. Antral infection was associated with increased severity in gastritis [6,7].

For research purpose modified Sydney system of classification is used wherein features as seen in endoscopy of gastric mucosa like erythema or exudation, erosion, mosaic pattern or cobble stone appearance, hypertrophic rugae, nodular and atrophic appearance are considered as abnormal. Erosive gastritis has been defined by white base lesions, either raised or flat, surrounded by a margin of intense erythema. Similarly unequivocal erythema or exudation, mosaic pattern, hypertrophic rugae , nodular and atrophic appearance in endoscopy are features suggestive of non erosive gastritis [8].

Non invasive methods of detection of Helicobacter pylori is with blood antibody test, carbon urea breath test, stool antigen test, urine ELISA test etc. Invasive methods include endoscopic biopsy and histological examination .When this is combined with either microbial culture or rapid urease test it is considered as one of the most reliable detection method [9]. The Rapid urease test detects Helicobacter pylori infection within an hour with an accuracy of 90% and is widely accepted to initiate the eradication therapy [10].

The determinants of infection particularly the socioeconomic standard of living and heterogeneity of infection even within a country and with different risk groups affects the success of a given therapeutic regimen. Increased rate of resistance to organisms and difference or even failure to respond pointed out the need to have a specific therapy regimen tailored for a particular region within a state or a country[2].

There is a requirement for prospective epidemiological data of high quality as emphasized in second Asia Pacific consensus guidelines for Helicobacter pylori infection(APCGH) especially from India[2]. This study aims at finding the relation of Helicobacter pylori infection with erosive and non erosive gastritis, also to evaluate the effects of the treatment with a fourteen day regimen of Amoxicillin, Metronidazole and Proton pump inhibitors in our set of population from South India.

AIMS & OBJECTIVES

1. To find out the relationship of Helicobacter pylori infection with erosive and non-erosive gastritis.

2. Effect of Anti Helicobacter pylori therapy on both types of gastritis.

3. To compare the effects of Anti Helicobacter pylori therapy on erosive and non-erosive gastritis.

REVIEW OF LITERATURE

Helicobacter pylori was linked to chronic gastritis and gastric ulcer following its discovery 1982.Previously they were not believed to be from a microbial cause. But since then many of the gastroenterological disorders were postulated to be caused by this organism. Even extra-gastrointestinal diseases have been researched for Helicobacter pylori as their cause. The organism is thought to be an essential component of the natural ecology of stomach. Even when more than half of the population harbours this organism only 20% of the individuals are causing disease or infection.

Helicobacter pylori though global, its more in the developing than in the developed countries. The sole source of the organism is the human gastric mucosa. The exact mechanism of transmission is not clear but it has been postulated to be or -oral or feco-oral .Poverty, over crowding and poor hygiene favours transmission and thus explains its increased prevalence in the developing countries.

Helicobacter pylori is a helical gram negative microaerophilic bacteria about 3 to 4 micrometres long and 0.5 micrometer in diameter .It produces oxidase, catalase ,urease and derive its energy from molecular

hydrogen with the help of hydrgenases. It has ability to from biofilms which help to avoid the acidic environment and various adaptations which adds to its survival, epidemiology and pathogenicity.

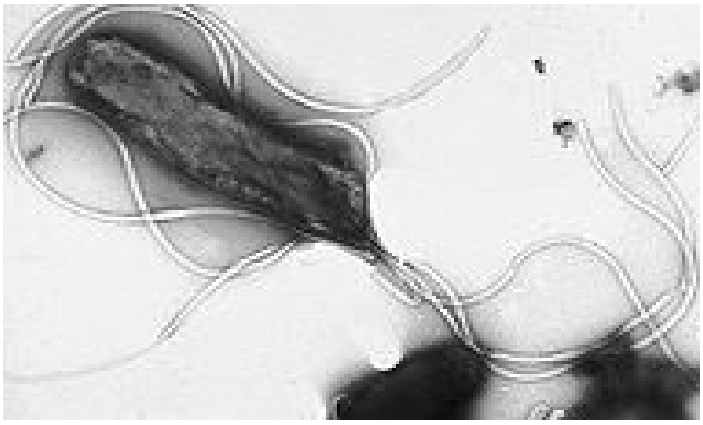


Fig 1. Helicobacter pylori with its lophotrichous flagellae

The bacterial structure consists of proteins like adhesions, porins, iron transporters, flagellum associated proteins. Being a gram negative organism they are composed of phospholipids and lipopolysaccharides. They have unipolar tuft of about four to six lophotrichous flagella and thus makes these organisms highly motile. Helicobacter pylori shows considerable genetic diversity as evident in its molecular typing. The complete genome of the bacterium has been mapped. The scientists have identified a 40 kilo base pair long Cag (cytotoxicity associated gene) pathogenicity island which has about 40 genes responsible for the pathogenicity of Helicobacter pylori. These island were absent in the Helicobacter pylori strains isolated from the

asymptomatic individuals. Virulence of Helicobacter pylori has been associated with certain alleles in genes like vac(vacoulating cytotoxin gene) in addition to the cag gene .

The genome study of Helicobacter pylori is under progress with increasing emphasis attempts to understand its pathogenicity and the various diseases for which it has been postulated as the cause.

PATHOPHYSIOLOGY

This bacteria belonging to phylum -Proteeobacteria, order-Campylobacterales and family Helicobacteraceae has a large diversity of strains. They have to tide over the extremely acidic environment of the gastrointestinal tract. It has various adaptations to avoid the acidic environment and burrows beneath the gastric mucosal lining where there is a neutral pH with the help of its lophotrichous flagella. They adhere to the cells with adhesins. By means of chemotaxis they avoid areas of acidic pH and they produce increased amounts of enzyme urease, which will break down urea to ammonia and carbon dioxide ,they neutralize the acidic environment. The ammonia being basic neutralizes the acid in the gastric mucosal lining of the stomach .

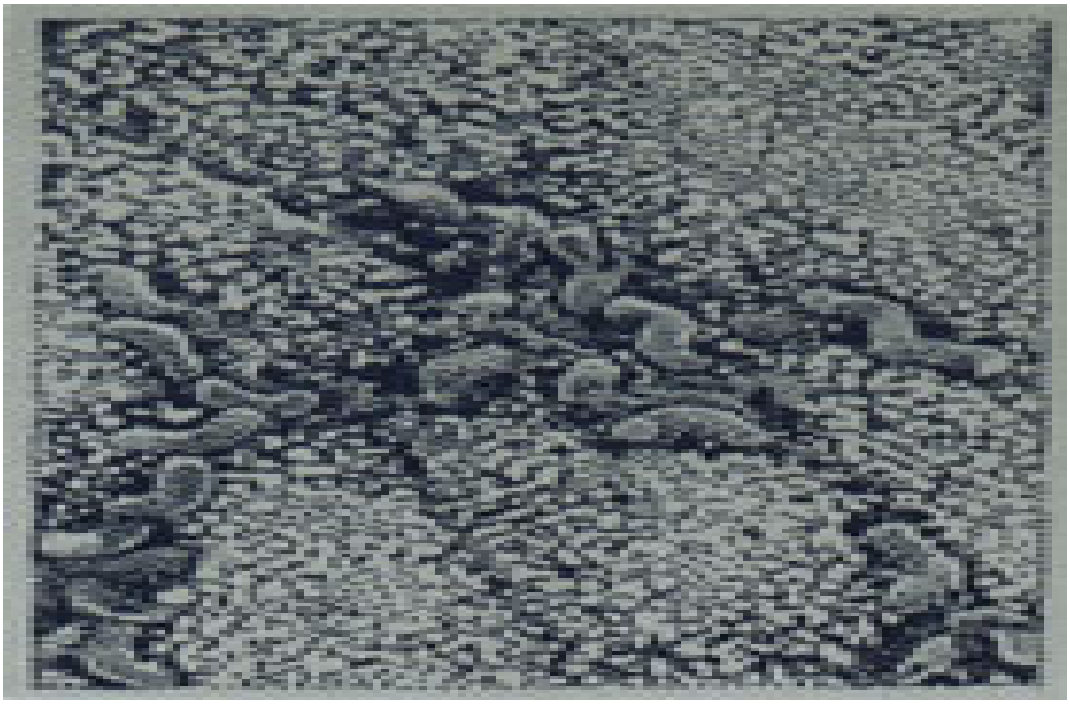


Fig 2. Helicobacter pylori as seen in scanning electron microscope

Certain subtle changes occur in the gastric mucosal lining due to several mechanisms during an infection. Firstly ammonia which is produced to neutralize the pH is toxic to the mucosal layer. Secondly proteases, vacoulating cytotoxin A (VacA) and phospholipases. Thirdly cytotoxin associated gene (CagA) causes inflammation and is potentially a carcinogen. These changes in the mucosa are used to predict the presence of the organism. The strains of Helicobacter pylori that produces high levels of VacA and CagA causes greater tissue damage and is evidenced in the endoscopic findings.

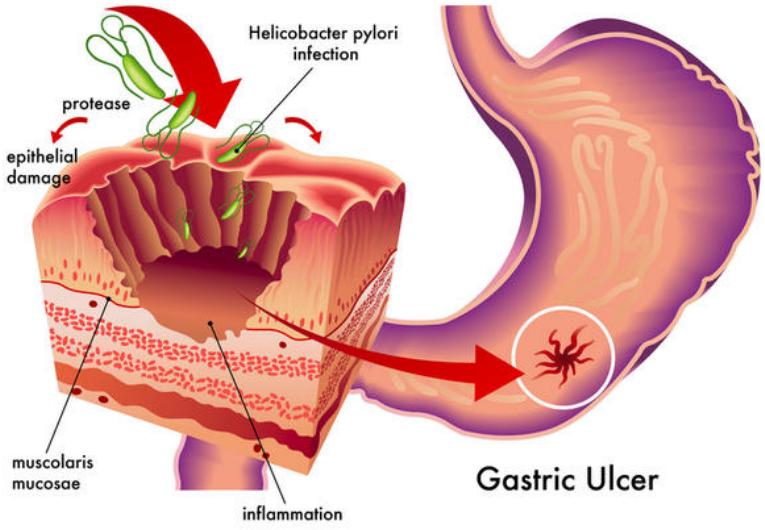


Fig 3 : Mechanism of H.pylori infection

Colonization of the gastric mucosa results in chronic gastritis. The Helicobacter cysteine rich (Hcp) proteins are a trigger to an immune response leading to inflammation. Thus ulcers of stomach and duodenum develops and the breach in gastric mucosal barrier will result in worsening of the symptoms .Further the G cells in the antrum produce gastrin in response to the inflammatory response which results in stimulation of the parietal cells to fill more acid into the gastric lumen thus forming a vicious cycle.

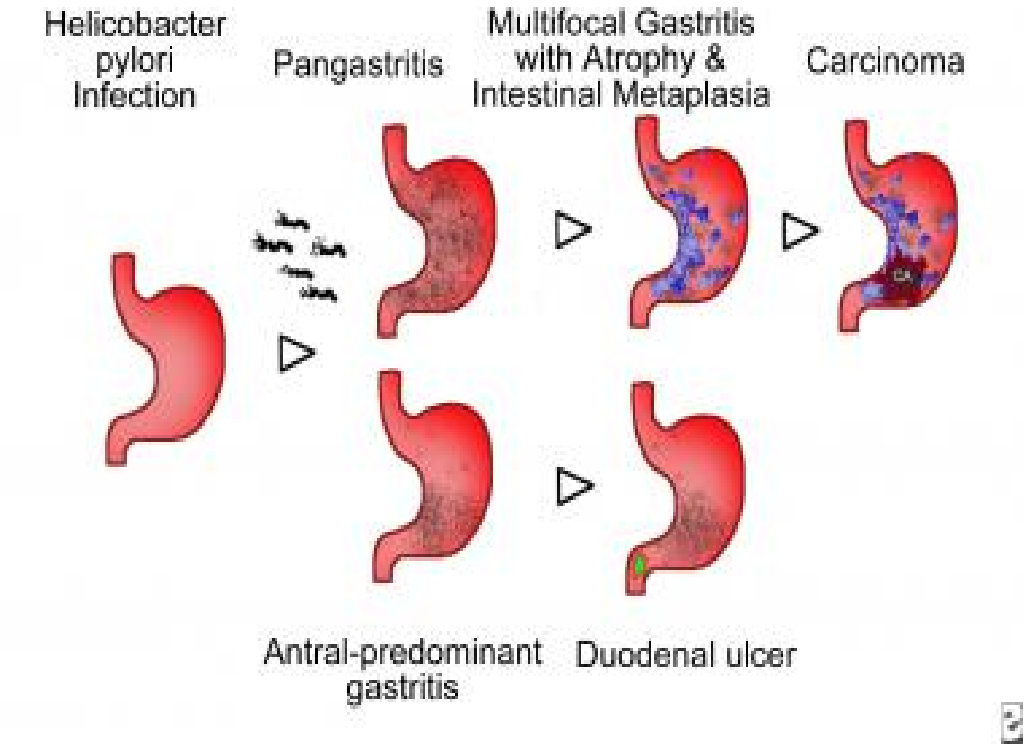


Fig 4 : Types and progression of H.pylori infection

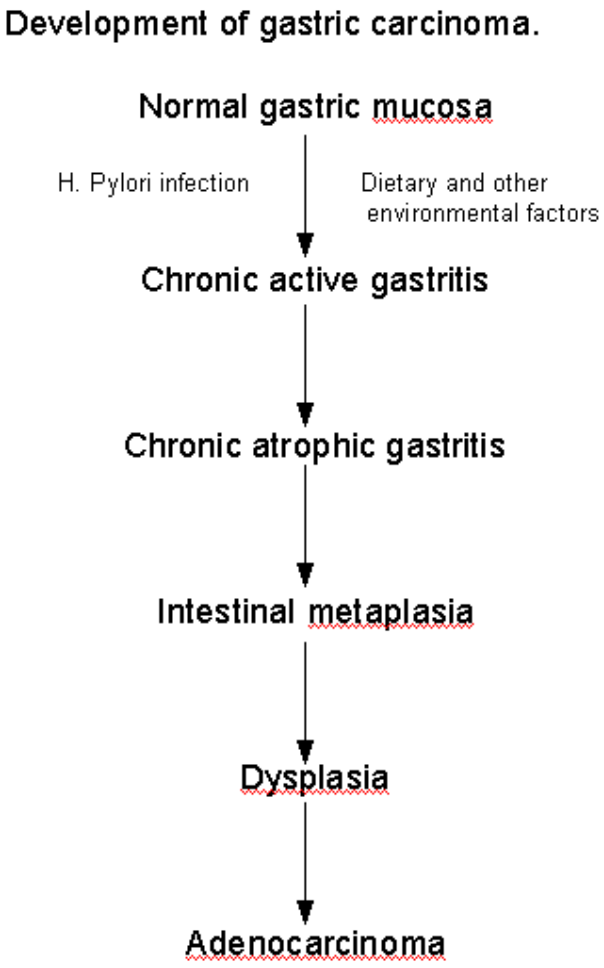


Fig 5: Flowchart showing sequence of development of cancer

Persistent inflammatory response induced by the bacteria will result in the atrophy of gastric mucosal lining and will result in gastric adenocarcinoma. The Mucosa associated lymphoid tissue(MALT) lymphomas are antigen driven and regress with elimination of the organism.Two mechanisms have been proposed by which the organism could cause cancer .It may involve the increased production of free radicals which results in increased rate of mutations to the host cells in the vicinity of Helicobacter pylori. The other mechanism involves alterations in the cell

adhesion proteins resulting in the enhancement of the transformed cell phenotype and this has been termed as “perigenetic pathway”.

SIGNS AND SYMPTOMATOLOGY

About 80% of the population infected with Helicobacter pylori is asymptomatic. Acute gastritis with abdominal pain or nausea may be the presenting feature[11]. When the disease progresses and develops chronic gastritis it causes severe abdominal pain, bloating, belching and vomiting. Dyspepsia which is defined as an abdominal or retrosternal chest pain is a major health problem. Gastroesophageal reflux disease(GERD) ,peptic ulcer disease and gastric carcinoma are usually associated with dyspepsia .Most common type of dyspepsia is functional dyspepsia and is defined as persistent abdominal and retrosternal discomfort in whom reasonable clinical evaluation has failed to reveal a definite cause for the symptoms [12,13]. Helicobacter pylori is proven to play a significant role in causing symptoms of functional dyspepsia.

Association of the organism in gastropathy associated with analgesics like NSAIDS(Non steroidal anti inflammatory drugs), gastroesophageal reflux disease and functional dypepsia are under research with the completed studies have proven the association [2] . Significant association with Portal hypertensive gastropathy in cirrhotic patients and its severity has been established[7]. Even association with extra-gastrointestinal disease has been established as in case of skin diseases like chronic urticaria, rosacea, psoriasis, Sjogrens syndrome lichen planus , behcets disease , systemic sclerosis etc [2,3] . Rarely, symptoms of all these diseases can occur in case of infection with Helicobacter pylori.

DIAGNOSIS

Testing for infection with Helicobacter pylori is recommended in case of peptic ulcer disease, a suspicion of low grade MALT lymphoma, after endoscopic resection of gastric carcinoma, in 1st degree relatives with gastric cancer patients. There are numerous tests available to detection of the organism. All the diagnostic tests available can be classified to two types either, invasive or non invasive [14].

Invasive tests : They involve the endoscopic biopsy of the gastric mucosa and examination of the sample with culture ,microscopy and urease tests. Microscopy of biopsy sections by silver staining or gram staining is useful. Other staining methods used are with Giemsa ,Haematoxylin –eosin ,acridine orange and even the use of a phase contrast microscopy. Culture is more sensitive but requires expertise and takes nearly three to seven days. Rapid urease test is useful in getting result within minutes.

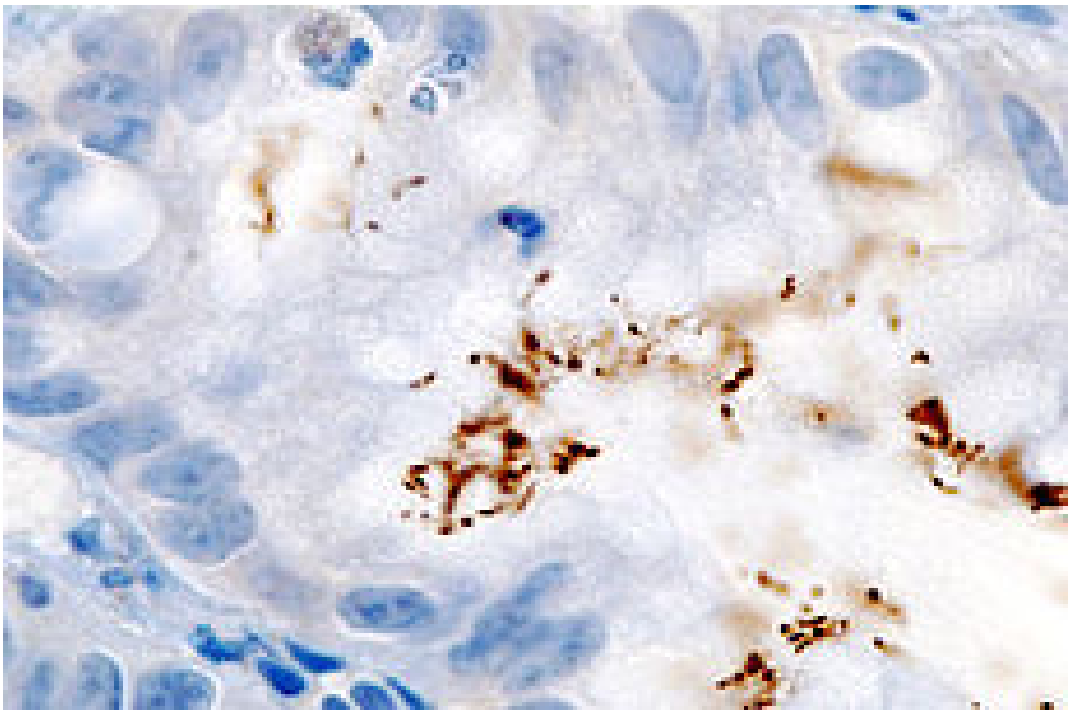


FIG 6 . IMMUNOHISTOCHEMICAL STAINING OF H.PYLORI GASTRITIS BIOPSY SPECIMEN

Non invasive tests include serology with ELISA and urease breath test. The urease breath test is sensitive and reliable but needs better isotope assay facilities.

The Second Asia- Pacific Consensus Guidelines for Helicobacter pylori infection (APCGH) has stated that when there is no need for an endoscopy ,C14 urease breath test is approved as an accurate non invasive test for the assessment of results and initial diagnosis of the anti Helicobacter pylori eradication treatment. But the test shows greater variability because of the non uniformity of the test parameters used in the various labs [2].C13 urea breath test is superior in this aspect. Breath tests had shown consistent high diagnostic accuracy among the non invasive tests and results were almost comparable with invasive tests like the biopsy based tests.

APCGH had recommended for outcome assessment after eradication treatment as there was high failure rate among significant proportion of population in terms of achieving eradication. Persistent undetected infection with Helicobacter pylori makes the patient prone to complications of infection .The importance of determining outcome will depend on the indication of treatment. Discordant opinion regarding need for assessment was there in the consensus but since the practice varies between countries it was agreed to customize according to that particular region. Re assessment of the infected patients should be done in 4 weeks following the completion of eradication treatment .It was suggested to withhold the proton pump inhibitors preferably one week before the post therapy assessment.

Least accurate diagnostic tests are the serological tests for Helicobacter pylori infection and are not that useful in determining the outcome of therapy as stated in Statement 14 of APCGH[15]. In case of bleeding patients the biopsy based tests may give a false negative value and in that case serological tests plays an important role. Also with the use of proton pump inhibitors and antibiotic use there is an increased false negative rate in biopsy, stool antigen and breath tests and in this scenario serological tests were proven to be useful.

Statement 3 of APCGH had emphasized that in Helicobacter pylori infective patients who have uninvestigated dyspepsia without alarm features the appropriate strategy would be to "Test and Treat". The statement 4 recommend that infection in case of Gastroesophageal reflux disease it is not recommended to test and treat the patient. But in case of an erosive esophagitis which was endoscopically proven it is advisable give the therapy because in Asia, peptic ulcer disease and gastric carcinoma are more common and there will be commensurately greater benefit. Section 6 of APCGH states that to screen and treat the Helicobacter pylori infection in populations with higher incidence of gastric carcinoma. This would effectively help in prevention of gastric cancer.

Prospective evidence of decrease in cancer was available in an eight year prospective study from China. The study had revealed that eradication in those who are in early stages and had not developed intestinal metaplasia and also patients with gastric atrophy had lower rate of gastric cancer when compared to those who were not on eradication therapy.

In the consensus there was agreement over the value of screen and treat strategy even in populations with low prevelance based on the new data which was available from various studies done in the last ten years. Also recent data from intervention studies showed a lesser risk for metachronous gastric cancer post eradication treatment and have thus emphasized the value of therapy in secondary prevention.

Thus even when the non invasive tests are considered to be better than the invasive tests according to the consensus guidelines, various factors taken into consideration proves the superiority of the invasive tests. Firstly the cost of non invasive tests like urea breath test limits its use in the developing countries .Secondly ,the use of invasive methods gives the added benefit of visualization of the changes in the gastric mucosa .

This is useful as more than giving the idea of the site of ulceration and classification of the gastritis as erosive and non erosive ,it helps in identifying any gastric atrophy or features suggestive of early gastric cancer . Thirdly the time required for diagnosis is less as rapid urease test just takes minutes to prove the presence of an infection. In case of severe infection the result is conclusive with in seconds but the non invasive tests takes days for diagnosis.

RAPID UREASE TEST

Rapid urease test is synonymous with CLO test (Campylobacter – like organism test) and has a high sensitivity and specificity and accurately (>90%) diagnose the infection .But active intestinal bleeding reduces the accuracy.

Principle of Rapid urease test

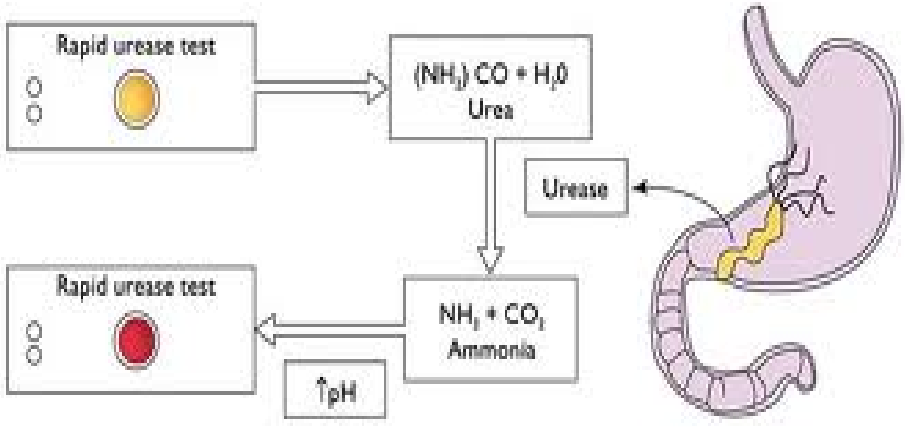


Fig 7 : Rapid Urease Test -mechanism

The diagram given below illustrates the steps to be taken for doinga Rapid Urease test(RUT).After endoscopically obtaining the sample from three different sites we have to follow these steps .

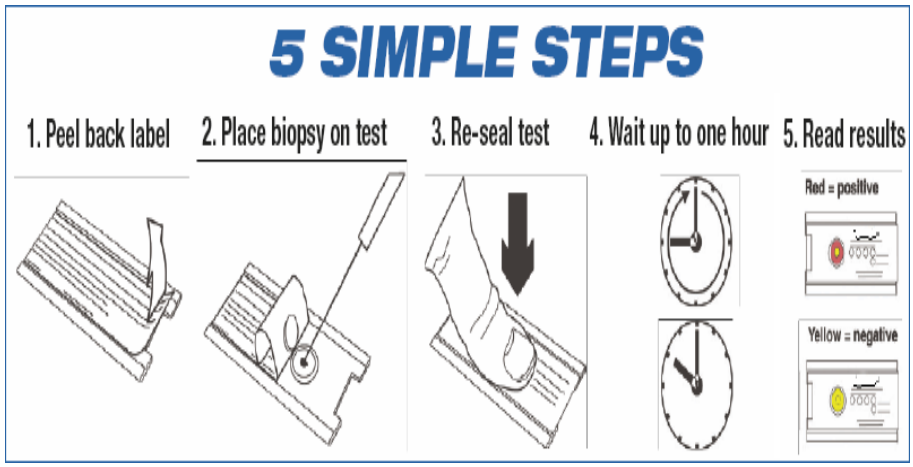


Fig 8: Steps of doing Rapid Urease Test

The interpretation is based on the colour which turns pink in the presence of the urease enzyme and in turn detects the Helicobacter pylori in the gastric biopsy sample.

Fig 9: Rapid Urease test results

TREATMENT

Various permutations and combinations of antibiotics, proton pump inhibitors along with other drugs like bismuth sulphate are used as therapy. Success of therapy depends primarily on the antibiotic sensitivity. Controversies over the use of various regimens have been addressed in various guidelines and consensus statements world wide like North America, Europe and the two editions of Asia –pacific consensus[10].

Before going into the treatment we will look upon some details which came in the 2nd Asia pacific consensus guidelines regarding the indications for treatment.

Indications for treatment

- Gastric ulcer disease (A)
- MALT Lymphoma (A)
- Atrophic gastritis (B)
- gastric cancer-post resection (B)
- Patients with 1st degree relatives having gastric cancer (B)

- Patients' choice (after consultation with their personal physician) (A)

- Non-ulcer dyspepsia (A)

- To decrease the risk of peptic ulcer and upper gastrointestinal bleeding in non-steroidal anti-inflammatory drug(NSAID) users (A)

- Before starting long-term aspirin therapy for patients with higher risk for ulceration and ulcer related complications (B)

- Patients on long-term low-dose aspirin therapy and who have a past history of upper gastrointestinal bleeding and perforation (B)

- GERD patients requiring long-term proton pump inhibitor (B)

- As a strategy for prevention of gastric cancer in communities with higher incidence of gastric carcinoma (A)

- Unexplained iron-deficiency anemia, or ITP(idiopathic thrombocytopenic purpura) (C)

Here A ,B and C represents the grade of recommendation

Statement 15 of APCGH states that in asia the currently recommended first line therapy for Helicobacter pylori is the use of a protonpump inhibitor with Amoxicillin and clarithromycin for a duration of seven days. Metronidazole has been considered as an acceptable alternative to amoxicillin or clarithromycin in the triple therapy regimens. In Asia where Amoxicillin is preferred metronidazole is less used.

In case of penicillin allergy metronidazole is considered the first line substitute .Another option is to start on bismuth based quadruple therapy as first line. As mentioned earlier various options can be worked out and selected as regimen to that particular area based on certain characteristics pertaining to the population in that area.

The various treatment regimens in treatment are discussed further

Treatment regimens for *Helicobacter pylori* as in 2nd APCGH

Triple therapy (Standard proton pump inhibitor therapy- (PPI)based):for 7–14 days

1. Amoxicillin 1 g, clarithromycin 500 mg with PPI twice daily

2. Metronidazole 400 mg, clarithromycin 500 mg with PPI twice daily

3. Amoxicillin 1 g, metronidazole 400 mg with PPI twice daily

Quadruple therapy for 7–14 days

Bismuth 240 mg twice daily, metronidazole 400 mg twice daily or three times daily, tetracycline 500 mg four times daily and PPI twice daily

Levofloxacin-based triple therapy for 10 days

levofloxacin 250 mg (or 500 mg), amoxicillin 1 g with PPI twice daily

Rifabutin-based triple therapy for 7–10 days

PPI, rifabutin 150 mg, amoxicillin 1 g twice daily

The second Asia Pacific consensus in its statement 15 had recommended first line therapy with Proton Pump Inhibitor(PPI) ,Amoxycillin and clarithromycin for 7 days[10]. Metronidazole was mentioned as an acceptable alternative to amoxicillin or clarithromycin.Further statements 16 and 17 in the consensus had highlighted about clarithromycin resistance and the fourteen day therapy[10]. Although the statement 17 states that the fourteen day therapy gives only limited advantage over seven day triple therapy regimen ,it highlights that out of the 21 studies used in the meta analysis only 2 were

Asian studies and none were Indian. A 5% increase in eradication rate with fourteen day regimen was addressed[10]. Salvage therapies in case of resistance were included in the consensus.

Section 20 of the 2nd APCGH has recommended various choices for salvage therapy:

1. Triple therapy which has not been earlier used in the patient
2. Bismuth based quadruple therapy
3. Levofloxacin based triple therapy
4. Rifabutin based triple therapy

Quadruple therapy as a salvage therapy is time tested and experience with it is more than for other salvage therapies which has been used worldwide . The determination of use of salvage therapy depends on various factors like antibiotic resistance in the given area,, previous treatment, drug availability and suggested to depend on the local prevalence of tuberculosis in view of the rifabutin use. For example ,levofloxacin-based triple therapy may be an excellent second line treatment in areas with low levofloxacin resistance. Rifabutin is considered only in tuberculosis low prevalence areas.

GASTRITIS

Gastritis is defined as the inflammation of the mucosal lining of the gastric region. There are very many causes leading to gastritis ,one of the most frequent causes is Helicobacter *pylori* infection and use of analgesics. Other causes include alcohol, smoking, drug abuse, critical illness, autoimmune conditions, radiation etc.

Majority of the population affected by gastritis are asymptomatic. But if symptomatic, abdominal pain is the most common presentation. It is described as a dull, vague, burning, aching, sore, or sharp type of pain which is localized to the upper central portion ,[11]but can be diffuse affecting any part of the abdomen or even around to the back.

Signs and symptoms associated with gastritis are listed below:

- Nausea
- Vomiting
- Bloating sensation
- Early satiety
- Appetite loss

- Unexplained weight loss

Classification of gastritis was first put forward by Whitehead in 1972 based of the mucosa type and severity of disease activity .This was followed by Strickland –Mackay classification and later the Glass classification. In 1990 Sydney classification of gastritis was approved .This classification was based on endoscopic findings, etiology ,site of ulceration etc

Based on the endoscopic findings it was classified into:

1. erythematous or exudative type
2. superficially erosive
3. polypoid gastritis with erosions
4. atrophic
5. hemorrhagic gastritis
6. bile gastritis
7. giant folds gastritis

Classification according to etiology

1. Autoimmune gastritis (type A)
2. Bacteria related gastritis (type B)
3. induced by Chemotoxic agents (type C)
4. distinct forms of gastritis

Other histological classifications are the Houston variation of Sydney system and the Operative Link for Gastritis assessment (OLGA) system which came in 2005.

Modified Sydney system of classification is where in endoscopic features of gastric mucosa like erythema or exudation, mosaic pattern or cobble stoning, erosion ,hypertrophic rugae, nodular and atrophic appearance are considered as abnormal and are used to classify gastritis It came into existence in 1996 and classifies gastritis into erosive and non erosive type.

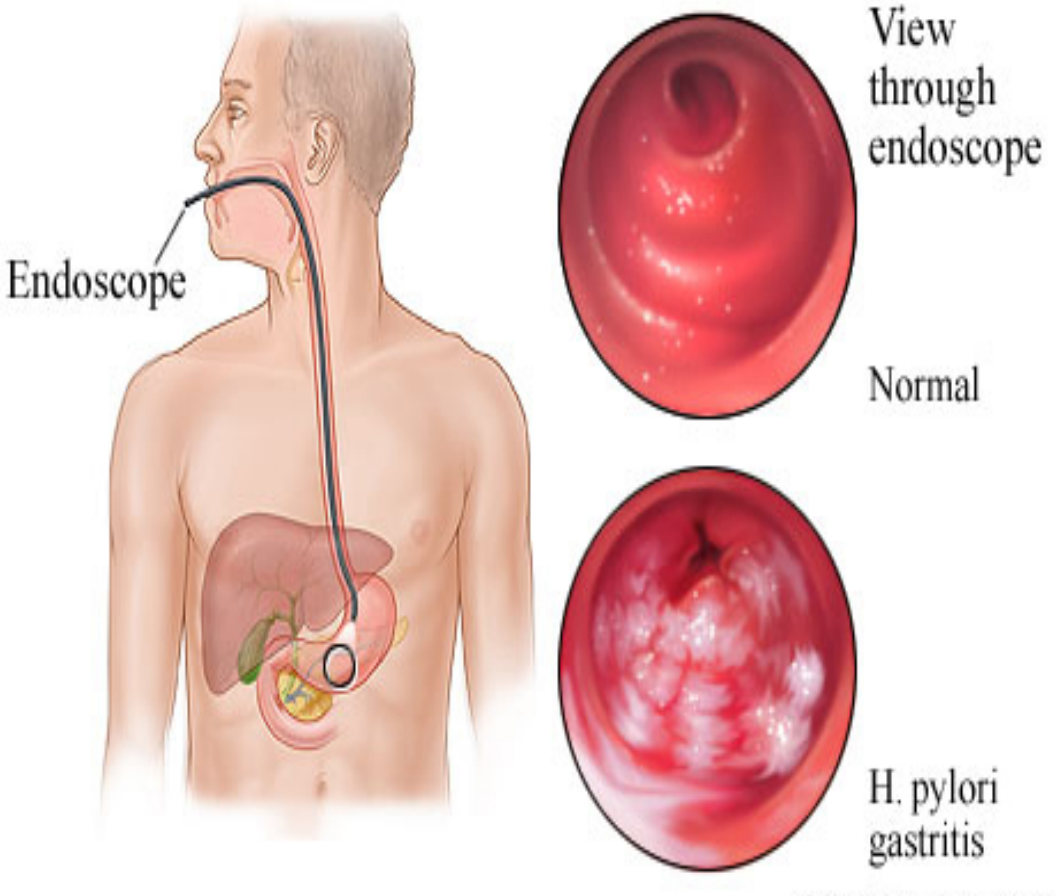


Fig 10 . Endoscopic findings

Erosive gastritis has been defined by raised or flat ,white base lesions, with intense erythema surrounding the margin. Similarly unequivocal erythema or exudation, hypertrophic rugae , mosaic pattern, atrophic or nodular appearance in endoscopy are suggestive features of non erosive gastritis.

Fig.11a. Non erosive gastritis

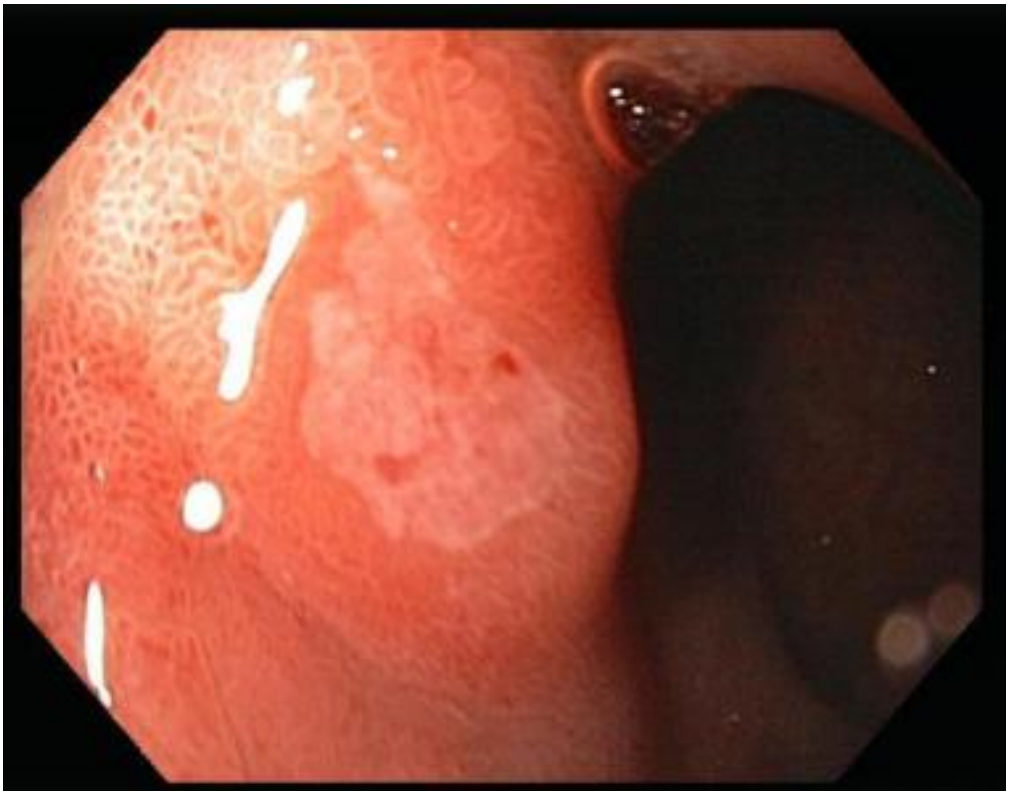


Fig11 b: Erosive gastritis

MATERIALS AND METHODOLOGY

PLACE OF THE STUDY

This study undertaken on the outpatients in the Gastroenterology department of Coimbatore Medical College Hospital ,Coimbatore.

PERIOD OF STUDY

July 2014 to July 2015.

DESIGN OF STUDY

Prospective study

METHODOLOGY

Patients aged between fifteen to sixty years having dyspeptic symptoms and willing to undergo for upper gastrointestinal endoscopy and anti Helicobacter pylori treatment were enrolled in this study. Every ethical issues which could arise were discussed with and informed written consent was taken from all patients.

After taking a detailed history and physical examination, patients were submitted to upper Gastrointestinal endoscopy and Rapid Urease Test(RUT) was done with one of the specimens taken from the predominant site of gastritis. Patients who were on Proton pump inhibitor or H2 blocker therapy were taken for endoscopy only after stopping these drugs for atleast 2 weeks. Repeat testing for checking the eradication was done four weeks after the completion of anti-*H. pylori* therapy.

Anti-*H. Pylori* therapy consisting of Metronidazole (500 mg bd) and Amoxycillin (1 gm) bd with omeprazole (20 mg bd)) for 14 days was used . Follow-up visits were made for assessment of compliance and side effects. After completion of therapy, clinical history was again taken and compared against the pretreatment symptoms. Frequency of infection to be calculated among endoscopically proven gastritis patients.

FOLLOW UP

Follow up visits were arranged for increasing compliance and for identifying any side effects. Follow up endoscopy was performed 4 weeks after completion of the therapy. Biopsy specimens were collected from antrum of stomach for the rapid urease test.

SELECTION CRITERIA

(a) INCLUSION CRITERIA

- Adults aged between 15 to 60 years having symptoms of dyspepsia.
- Those willing to undergo upper gastrointestinal endoscopy and anti-Helicobacter pylori therapy.

(b) EXCLUSION CRITERIA

- Patients who were regular users of NSAID and steroids, had peptic ulcer and its complications.
- Patients with history of previous Helicobacter pylori eradication therapy.
- Patients with coexisting gastric cancer, pregnancy or lactation and concomitant other severe diseases.
- Any acute bleeding episodes

SOURCE OF DATA

Data consists of primary data collected by the principal investigator directly from the patients who had approached Government Medical College Hospital,Coimbatore. The subjects consists of outpatients attending the Gastroenterology Outpatient Department.

STATISTICAL ANALYSIS

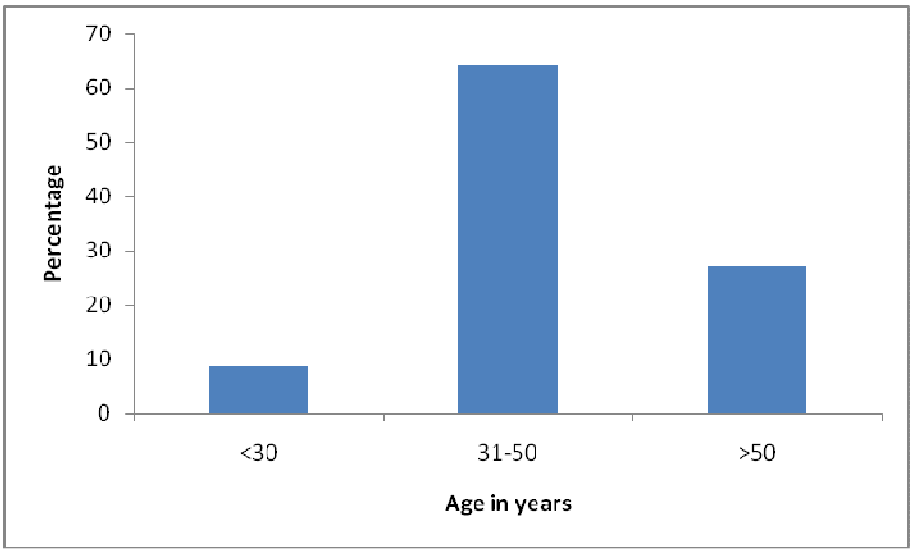
Clinical features, symptoms, endoscopic findings, pre- and post treatment disease status were compared with the help of. SPSS (Statistical Package for Social Services) 16.0 for statistical calculations.

OBSERVATIONS AND RESULTS

- Total of 92 patients were enrolled in the study, who presented with dyspeptic symptoms and were willing for the study between the period of July 2004 to July 2015.
- 81 patients out of the 92 enrolled had endoscopically proven gastritis.
- Out of the 81 endoscopically proven gastritis patients, 72 patients were proven to be infected with helicobacter pylori based on the Rapid Urease test.
- 72 patients who were H.pylori infected were treated and were followed up and planned for repeat testing after 4 weeks of therapy.
- Out of the 72 only 48 patients could be successfully called back for a repeat testing following the therapy .Dropout percentage was 33%.(24 out of 72).
- Further studies and comparison were done with these 48 patieints.

AGE DISTRIBUTION

FIG 12. AGE WISE DISTRIBUTION OF GASTRITIS PATIENTS



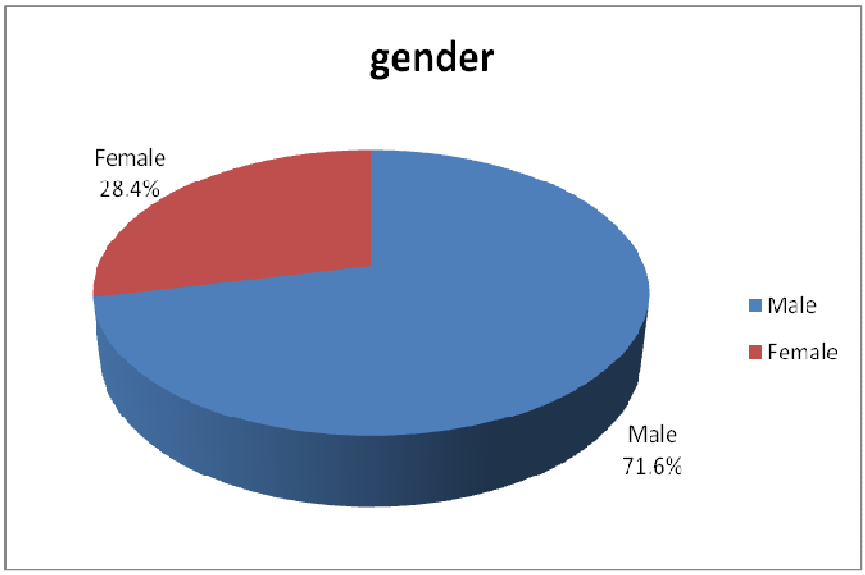
- Endoscopically proven gastritis changes in the mucosa were more in the age middle age group with about 64.2% (52) of the study population.
- About 1/3rd were of age group above 50 years.
- Mean age for males was 36 and that of females was 38

SEX DISTRIBUTION

Male : Female ratio was nearly 3:1

58 males(71.6%) : 23 females (28.4%)

FIG 13: PIE CHART SHOWING THE GENDER DISTRIBUTION



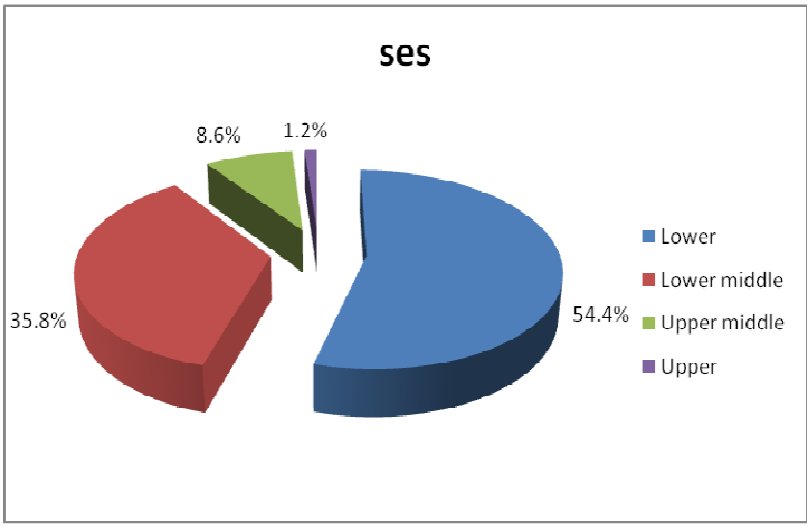
SOCIO ECONOMIC STATUS (SES)

Being a tertiary care centre in the government setup we had more than half of the study population coming from the lower socio economic strata(54.3%).

TABLE 1. SOCIOECONOMIC STATUS DISTRIBUTION

SES	Frequency	Percent
Lower	44	54.3
Lower middle	29	35.8
Upper middle	7	8.6
Upper	1	1.2
Total	81	100.0

FIG14. PIE CHART SHOWING SOCIOECONOMIC STATUS DISTRIBUTION

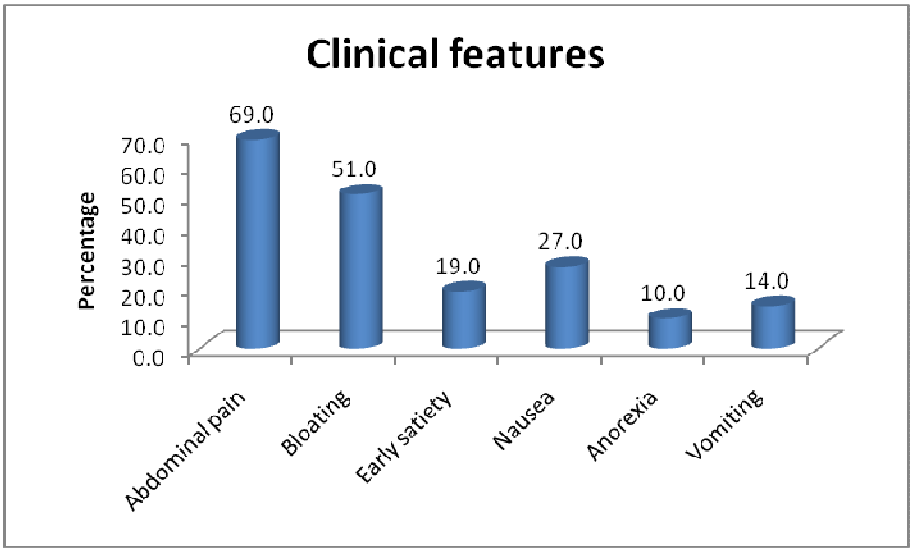


Thus lower middle and lower socio economic class had higher prevalence of gastritis among the study group (90.1%).

SYMPTOMATOLOGY

This histogram shows the various symptoms and their frequency in gastritis patients.

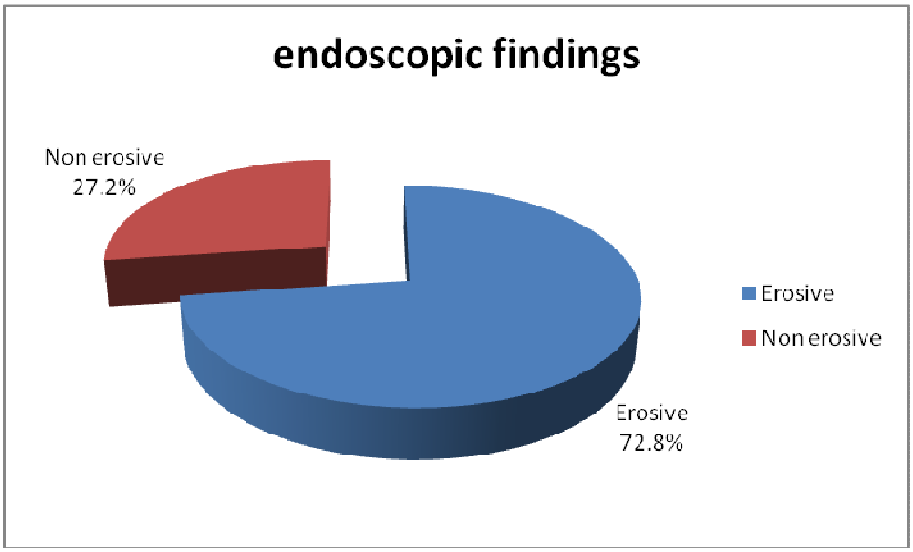
FIG 15. HISTOGRAM SHOWING FREQUENCY OF THE PRESENTING SYMPTOMS



- Abdominal pain is the commonest symptom (69%) followed by bloating sensation(51%).
- As in literature abdominal pain which is due to gastric mucosal damage in gastitis is the most common symptom.

ENDOSCOPIC FINDINGS

FIG 16: PIE CHART SHOWING ENDOSCOPIC FINDING DISTRIBUTION

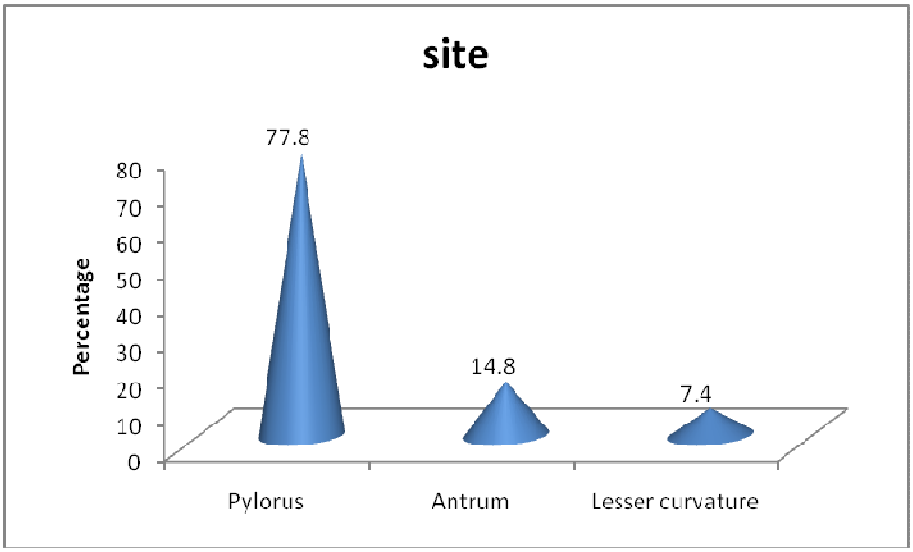


About 3/4th of the gastritis patients presented with erosive type of gastritis which causes severe symptoms and also persistent infection ,if left untreated leads to the complications.

Out of the 81 endoscopically proven gastritis patient 59 (72.8%)had erosive type and 22(27.2%) had non erosive gastritis.

SITE OF LESION

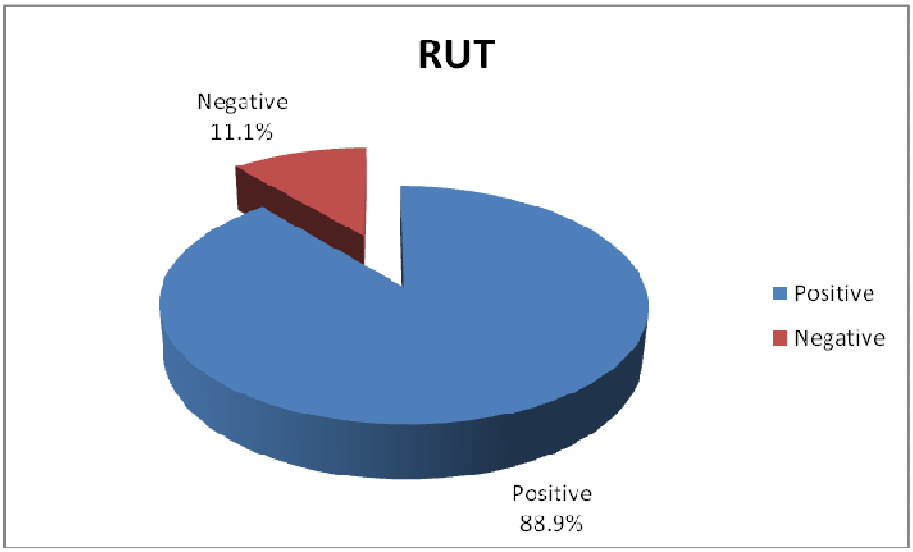
FIG 16 . CONE HISTOGRAM SHOWING DISTRIBUTION OF THE SITE OF GASTRITIS



- Major site of infection leading to gastiritis was at pylorus (77.8%) and least involved region was the lesser curvature (7.4%).
- About 10 times more prediliction for the pyloric region to be affected than the lesser curvature

RAPID UREASE TEST

FIG 17 : PIE CHART SHOWS THE DISTRIBUTION OF THE RAPID UREASE TEST



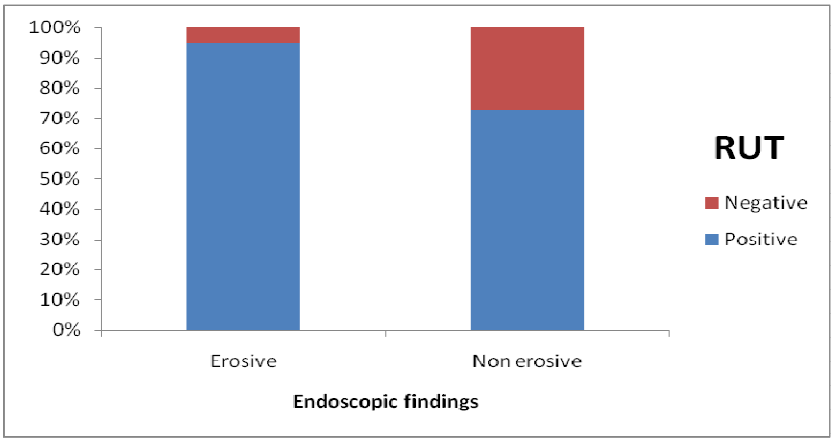
- Out of the 81 endoscopically proven gastritis patients 72 (88.9%) had Rapid urease test positivity .
- Prevalence of Helicobacter pylori infection in endoscopically proven gastritis patients were as 88.9%.
- Thus our part of country is a high prevalence area.

RUT & TYPE OF GASTRITIS

TABLE 2: TABLE SHOWING RELATIONSHIP BETWEEN RUT & GASTRITIS

RUT	Endoscopic Findings			
	Erosive		Non erosive	
	N	%	N	%
Positive	56	94.9	16	72.7
Negative	3	5.1	6	27.3
Total	**59**	**100**	**22**	**100**

FIG 18: HISTOGRAM SHOWING RELATIONSHIP BETWEEN RUT & GASTRITIS

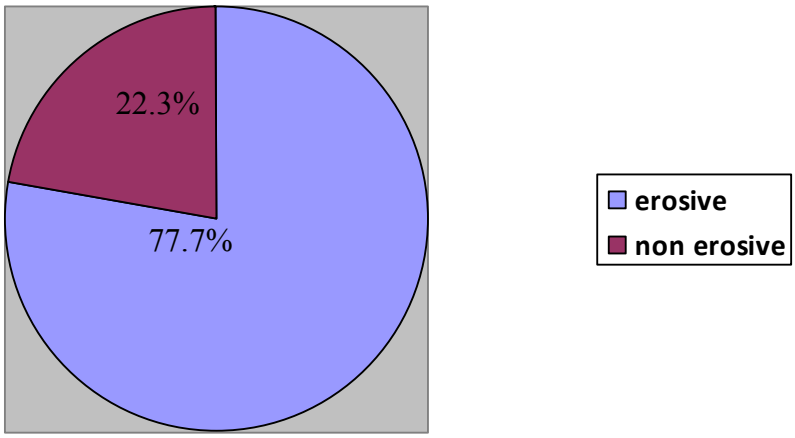


df=1 p=0.015

Among the 59 erosive gastritis patients 56 (95%) had H.pylori infection and in case of the 22 non erosive gastritis patients 16(73%) had H.pylori infection

Among the 72 H.pylori positive patients 56 (77.7%) had erosive gastritis and 16 (22.3%) had non erosive gastritis.

FIG 20: SHOWS RELATIONSHIP BETWEEN H.PYLORI INFECTION AND TYPE OF GASTRITIS



FOLLOW UP PATIENTS

- Out of the 72 H.pylori infected patients who were instituted therapy 48 had came for post therapy testing to assess eradication.

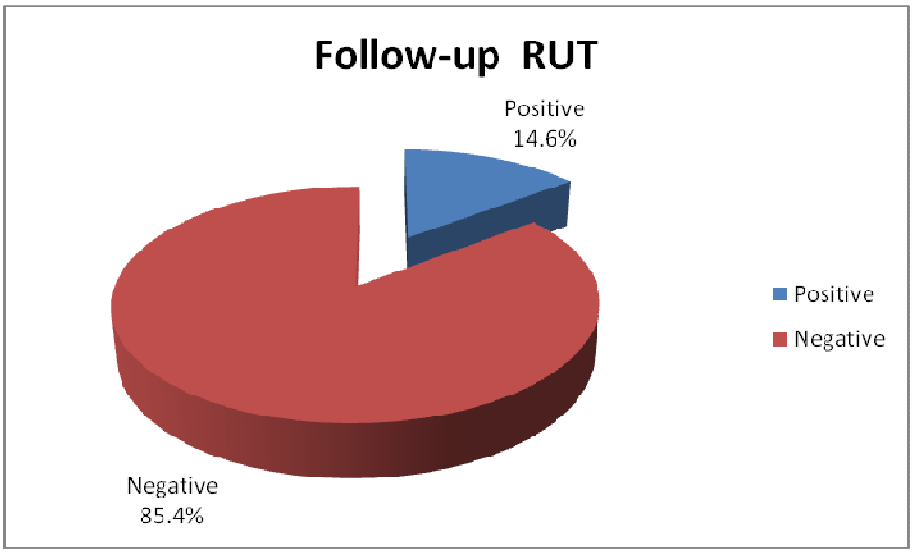
- 37 of the 48 were from the erosive group and 11 were from the non erosive group

TABLE 3: FOLLOW UP PATIENTS & THE GASTRITIS TYPE THEY BELONGED

Follow up patients	Frequency	Percent
Erosive	37	77.1
Non erosive	11	22.9
Total	**48**	**48**

FOLLOW UP RAPID UREASE TEST

FIG 21: PIE CHART SHOWING FOLLOW UP RAPID UREASE TEST



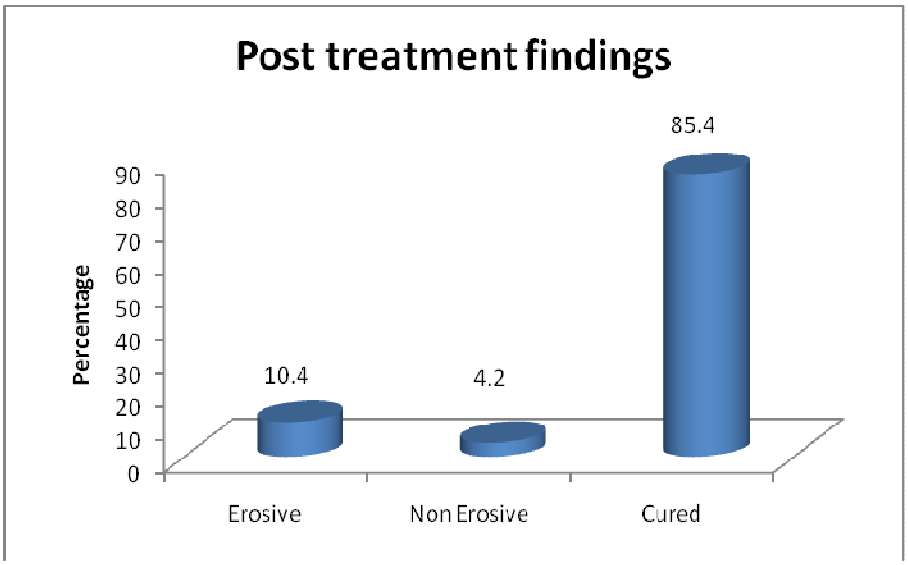
Out of the 48 patients who were followed up in the study 41 became RUT negative(85.4%).Thus the eradication rate in our study was nearly 86%.

POST TREATMENT ENDOSCOPIC FINDINGS

TABLE 4: ENDOSCOPIC FINDINGS AFTER TREATMENT

Post treatment findings	Frequency	Percent
Erosive	5	10.4
Non Erosive	2	4.2
Cured	41	85.4
Total	**48**	**100.0**

- Post treatment endoscopic findings revealed a cure rate of 85.4% (41/48) with about 15% of patients having persistent gastritis.
- Of the uncured 70% (5) were erosive and 30%(2) were non erosive.



CLINICAL SYMPTOMS POST TREATMENT

TABLE 5: TABLE SHOWING CLINICAL SYMPTOMS POST TREATMENT

Clinical features	Before treatment		After treatment			
	N	%	Resolved		Not resolved	
			N	%	N	%
Abdominal pain	36	75.0	25	52.1	11	22.9
Bloating	26	54.2	18	37.5	8	16.7
Early satiety	12	25.0	8	16.7	4	8.3
Nausea	10	20.8	8	16.7	2	4.2
Anorexia	5	10.4	5	10.4	0	.0
Vomiting	7	14.6	7	14.6	0	.0

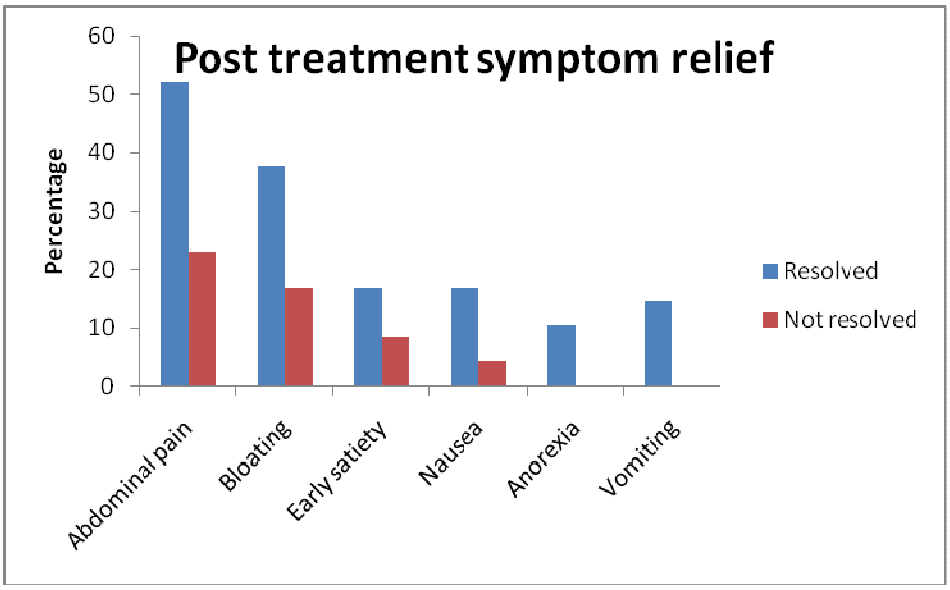
- There was drastic reduction in the clinical symptoms with abdominal symptoms nearly getting 50% resolved .

- Also other symptoms of bloating ,early satiety and nausea showed more than 30% resolution.

- There was total absence of symptoms of anorexia and vomiting in patients post therapy.

The effective resolution of symptoms in case of Helicobacter pylori infection in gastritis patients can be better understood with help of this histogram shown.

FIG 22: HISTOGRAM SHOWING RESOLUTION OF SYMPTOMS



ENDOSCOPIC RESOLUTION POST THERAPY

TABLE 6. COMPARISON OF ENDOSCOPIC FINDING PRE AND POST TREATMENT

	Post treatment						Pre treatment Endoscopic findings	
	Erosive		Non Erosive		Cured			
	N	%	N	%	N	%	N	%
Erosive	5	100	0	0	32	78	37	77.1
Non erosive	0	0	2	100	9	22	11	22.9
Total	5	100	2	100	41	100	48	100

- Out of the 37 erosive gastritis patients who had came for followup 32 patients had endoscopic clearance of the lesions following therapy and only 5 had persistent lesions.Of the 22 non erosive patients only 2 had persistent non erosive gastritis.

- Thus endoscopic cure rate was 86.4% in case of erosive gastritis and 81.8% in case of non erosive gastritis.

HELICOBACTER PYLORI ERADICATION

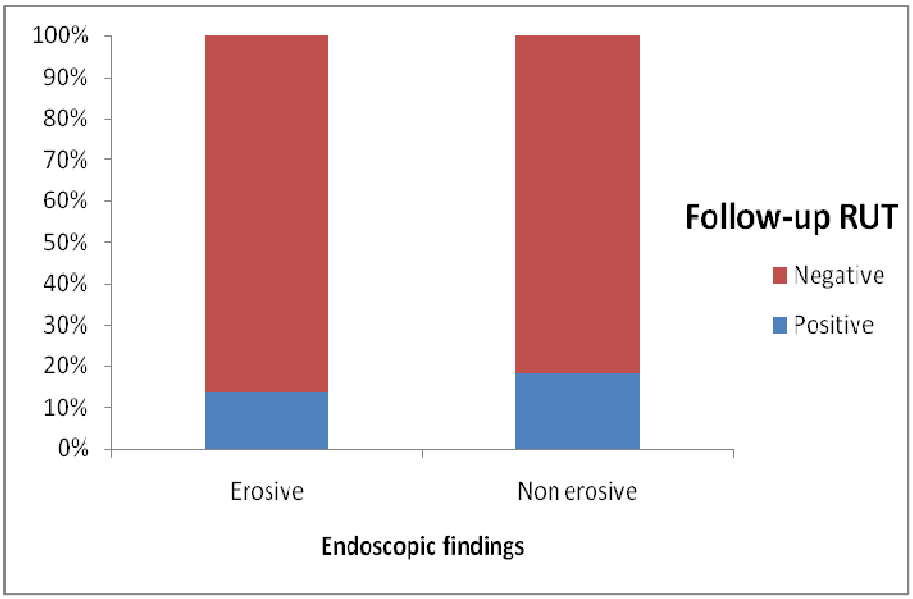
TABLE 7. SHOWING H.PYLORI ERADICATION RATE & GASTRITIS TYPES

Endoscopic findings	Follow-up RUT				Total	
	Positive		Negative			
	N	%	N	%	N	%
Erosive	5	13.5	32	86.5	37	100.0
Non erosive	2	18.2	9	81.8	11	100.0
Total	7	14.6	41	85.4	48	100.0

$\chi^2 = 0.000$ df=1 p=1.000

- Eradication rate for both erosive and non erosive gastritis patients infected with Helicobacter pylori were comparable and p value was not significant.
- Similar to the endoscopic cure rate ,the eradication rate was also 86.4% in case of erosive gastritis and 81.8% in case of non erosive gastritis.

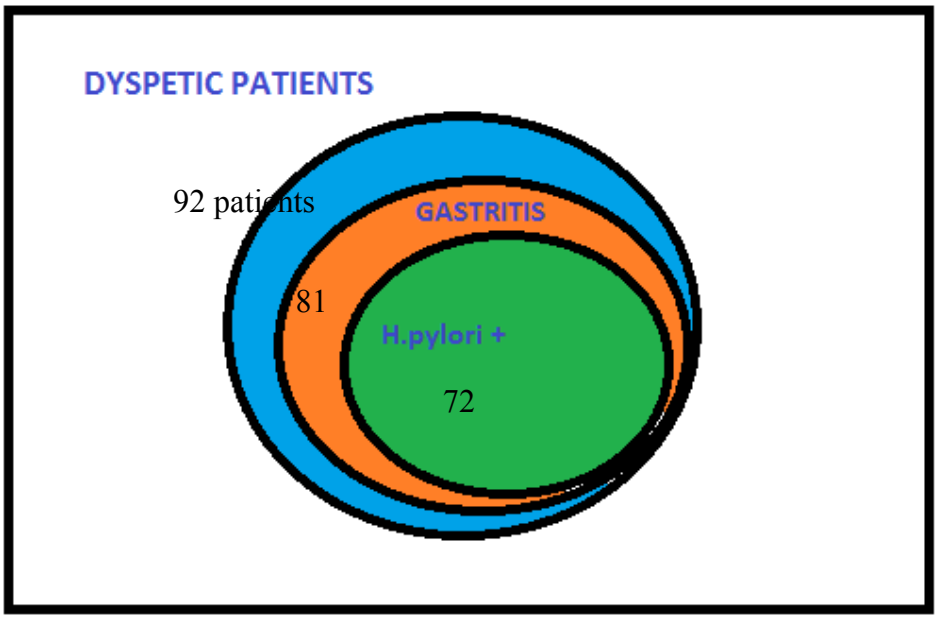
FIG 23: COMPARISON OF FOLLOW UP RUT WITH TYPE OF GASTRITIS



- This Histogram shows that the eradication rate was equally good in both erosive and non erosive gastritis but definitely better in erosive cases.

DISCUSSION

In our study, a total of 92 patients had fulfilled the inclusion criteria and were enrolled in the study. These were mainly the dyspeptic patients who had attended our gastroenterology out patient department and who were willing for the study which was done between the time period of July 2004 to July 2015.



81 patients out of the 92 enrolled had endoscopically proven gastritis. Out of the 81 endoscopically proven gastritis patients, 72 patients were proven to be infected with helicobacter pylori based on the Rapid Urease test.

Seventy two patients who were Helicobacter pylori infected were treated and were followed up and planned for repeat testing after 4 weeks of therapy.

Out of the 72 only 48 patients could be successfully called back for a repeat testing following the therapy .

Further study and comparison were done with these 48 patients to come to a conclusion.

Helicobacter *pylori* eradication in people infected results in reduction of gastric atrophy and gastritis. Subsequently the malignancy risk is drastically reduced with even near total cure of the low grade MALT lymphomas [9] .

AGE DISTRIBUTION

Our study population were mainly from the middle age group who were about 64.2% (52) of the total study population and one third were of age group above 50 years. This finding was similar to the world wide studies and WHO had stated that infection rate was more in middle age groups of 25 to50 years age when compared to other age groups.

SEX

There was a male preponderance in our study group owing to socio-economic status, literacy, awareness among females and decreased accessibility to health care as they are decreased to household activities.In our study there were 58 males(71.6%) and 23 females (28.4%) with a male to female ratio as high as 3 is to 1.

SOCIOECONOMIC STATUS

More than half of the study population were from the lower socio economic strata(54.3%).Socio economic status is significant because there is increased colonization in the poor socioeconomic status group and those with lesser education apart from considering the genetic factors in case of developing and underdeveloped countries.

The results from our study are in agreeing with the proven results of other studies done around the world which were determined by the place of the study,socio-economic status as well as the mode of transmission which causes the spread infection from person to person or by oro-oral or the feco-oral routes.

SITE OF INFECTION

In most persons, *H. pylori* infection is gastric antrum is the most affected site. Studies have shown that *H. pylori* infection occurs in the antrum in about 85% of patients with the disease, and in about 15% of patients orpus is the most affected site.11,12 Our study found out that nearly 78% of RUT positive gastritis were seen in the pylorus and 11.8% in the antrum of gastric mucosa.

ENDOSCOPIC FINDINGS BEFORE TREATMENT

Erythema as an endoscopic finding are frequently labeled as gastritis despite a lack of evidence supporting a correlation between endoscopic features and histologic gastritis.14

In our study, we found out that 77.7% had erosive gastritis and 22.3% non-erosive gastirits among the Rapid urease test positive Helicobacter *pylori* gastritis. Erosive gastritis has been defined by raised or flat ,white base lesions, with intense erythema surrounding the margin. Similarly unequivocal erythema or exudation, hypertrophic rugae , mosaic pattern, atrophic or nodular appearance in endoscopy are suggestive features of non erosive

gastritis.About 40% or more of patients have a false negative endoscopic reporting with endoscopically normal mucosa having histological gastritis visible in a biopsy specimen [18] . Almost always corresponding histologic inflammatory changes are present when the endoscopic changes are more pronounced and in case of erosions or frank atrophic gastritis . So generalizing the concept a more severe the endoscopic gastric defect, the better the correlation with the histology report from the biopsy[15,16] .

Our study had incorporated the Sydney system- endoscopic appearance of gastritis for diagnosis of gastritis without taking into consideration the histology. Khan et al observed that the erythematous gastritis was the single most common endoscopic finding in gastritis and Helicobacter *pylori* was detected in 74% of the patients. Stolte and Edit stated that antral erosion was a sequelae of Helicobacter pylori infection and that these chronic erosions caused by the organism in future would be differentiated from other erosions [19] .

INFECTION RATE

H. pylori had a strong association with gastritis2 which has been proven in various studies done worldwide. In this study, *H. pylori* infection rate in gastritis was 88.8%.

SYMPTOMATOLOGY PRE &POST THERAPY

Regarding symptomatology , abdominal pain is the commonest symptom (69%) followed by bloating sensation(51%) in our study population.Pre-treatment symptoms of abdominal pain, bloating sensation ,early satiety, nausea ,anorexia and vomiting had decreased from 75%,54.2%,25%,20.8%,10.4%, 14.6% respectively in the study population to a frequency of 22.9%,16.7%,8.3%,4.2% ,0%,0% respectively in the follow up patients post eradication therapy.Post therapy patients were completely relieved of symtoms of anorexia and vomiting.

In eradication studies18 there is an ongoing debate on whether dyspeptic symptoms decrease with anti-*H. pylori* treatment. This is explained with possibility of the high placebo-response rate and also that many therapy regimens have failed to cure the infection. Slowing of bacterial growth cannot affect symptoms of gastritis significantly, if these are due to mucosal inflammation, and symptom resolution may take many weeks or months following the eradication of Helicobacter *pylori* and the associated gastritis.

But our study proves the other studies wrong and shows that in areas of high prevalence eradication will result in more symptom relief as evidenced by resolution of abdominal pain, bloating sensation, early satiety, nausea, anorexia and vomiting in 52.1%, 37.5%, 16.7%, 16.7%, 10.4% and 14.6% patients respectively after treatment. Hence out treatment of the triple drug regimen of antibiotics and proton pump inhibitors were effective in providing symptomatic relief and also improved the patient compliance.

DETECTION WITH RUT

According to the Maastricht III consensus conference held in 2005, there was a recommendation that diagnosis can be confirmed and treatment can be started if Rapid urease test is positive [29] . There are a numerous RUT kits which are commercially available with an overall pretreatment sensitivities of >90% and specificities of >95%19 and these are sufficient enough to prove its use as a single test for Helicobacter pylori detection..

The Rapid urease test being simple, cost effective, and quick in providing results makes it a practical and economic means of testing for *H. pylori* infections in patients not taking antibiotics or proton pump inhibitors who need an upper Gastrointestinal endoscopy. Hence RUT was used as single best test for diagnosis of *H. pylori* gastritis in our study. As there is resolution of infection and as the distribution of *H. pylori* infection becomes patchy after antibiotics or proton pump inhibitors, biopsy for the Rapid urease test should be taken from two sites, the body and the antrum at area of greater curvature [30] . Due to limitation in facilities, we took biopsy from only predominant site of gastritis before treatment and only from one site after treatment for diagnosis and assessment of eradication .

In this study, Helicobacter pylori infection status was considered to be positive by a positive RUT test result. Based on this criterion, out of 81 endoscopically proven gastritis patients, 88.8% had *H. pylori* gastritis. The remaining 11.2% patients were negative for Rapid urease test.

Non-invasive tests can be employed for confirming the eradication of Helicobacter pylori like urea breath test or stool antigen test except in patients where repeat endoscopy is indicated, as in case of patients with gastric ulcer.19 As post therapy endoscopy was performed to identify the changes of gastric mucosa after triple therapy, Rapid urease tests were done for confirmation of the eradication of bacteria.

The treatment of Helicobacter pylori infection is a challenging clinical problem due to the increase in antimicrobial resistance and decreasing eradication rates. The third Maastricht Consensus Report agreed that effective treatment for *H. pylori* should achieve an intention-to-treat (ITT) eradication rate of over 80%. [6]

In clinical practice eradication rates are lesser than 80% for many of the standard therapy regimes. Lots of factors such as duration of therapy, type and combinations of the antibiotics and other supportive drugs used, ,more patient compliance and awareness may help to improve the rate of eradication of the bacteria[6,7] .

CURE RATE and TYPE OF GASTRITIS

Our treatment regimen with PPI-amoxycillin-metronidazole for 14 days was used in this study. This regimen was chosen as bulk of our patients came from lower or lower middle class of the society. Out of the 48 infected patients 41 were Rapid urease test negative which confirmed the eradication. So the eradication rate from the study was 85.4%. In clinical trials using anti-*H. pylori* treatment, the global eradication rate was only 64%.21 .

Out of the 37 erosive gastritis patients who had came for follow up 32 patients had endoscopic clearance of the lesions following therapy and only 5 had persistent lesions. Of the 11 non erosive patients only 2 had persistent non erosive gastritis.

Thus endoscopic cure rate was 86.4% in case of erosive gastritis and 81.8% in case of non erosive gastritis. In our study the eradication rate of Helicobacter pylori and the endoscopic cure rate were going hand in hand.

In the present study, strong relationship between *H. pylori* infection and gastritis was found. Majority of cases had pyloric erosive gastritis. After treatment with *H. pylori* eradication therapy, significant improvement of endoscopic feature of gastritis occurred and the erosive group responded only slightly better than non-erosive group. But what was significant in this study was that both groups showed a very high response rate in terms of endoscopic clearance of lesion and in terms of eradication of Helicobacter pylori.

SUMMARY

Prevalence of Helicobacter pylori infection among endoscopically proven gastritis patients in our study was nearly 89%. Hence proves that our part of country is a high prevalence zone after taking into consideration the effect of hospital bias.

High prevalence could be also due to the socioeconomic status. Our study had 54.3% belonging to lower class and 35.8% from the lower middle class. Thus about 90% of the study population were from low income group and proves the higher rate of infection among the lower socioeconomic groups.

Endoscopically proven gastritis changes in the mucosa were more in the age middle age group with about 64.2% (52) of the study population.

Abdominal pain is the commonest symptom (69%) followed by bloating sensation(51%) in case of an infection which was proven in other studies.

Among the 72 H.pylori positive patients 56 (77.7%) had erosive gastritis and 16 (22.3%) had non erosive gastritis. Shows the increased association H.pylori infection with erosive gastritis.

Dropout rate in our study was just 33% .About 67% of study population had returned for post therapy evaluation

Out of the 48 patients who were followed up in the study 41 became RUT negative(85.4%).Thus the eradication rate in our study was about 86%.This denotes the effectiveness of the Standard triple therapy instituted in our tertiary care centre. This was high when compared to H.pylori eradication studies done world wide.

Thus endoscopic cure rate was 86.4% in case of erosive gastritis and 81.8% in case of non erosive gastritis. In our study the eradication rate of Helicobacter pylori and the endoscopic cure rate were going hand in hand.

Even though the cure rate in both types of gastritis were high there was no statistically significant difference in eradication rate or endoscopic resolution of lesion in erosive and non erosive types.

So effective treatment and follow up of patients with gastritis will improve eradication of the organism and prevent complications.

CONCLUSION

Our part of country is a high prevalence zone of Helicobacter pylori infection as proven with 89% positive cases among gastritis patients in our study.Also this increased prevalence could be attributed to the low socioeconomic status as majority of our study population were lower socioeconomic group. Since this infection affects the middle age population the most, who are the bread winners and this in turn affects the quality of life and economic status forming a vicious cycle.

Early detection and institution of eradication therapy is the need of the hour. Our study concludes that gastritis has strong association with Helicobacter pylori infection and with triple therapy we follow we have the highest eradication rate of about 86% both in case of erosive(87%) as well as non erosive gastritis(82%).There was no significant difference in cure rate in the two types of gastritis.

Along with eradication of Helicobacter pylori there is drastic improvement of the symptoms as well as increased endoscopic clearance of the lesion. The increased symptom relief in these patients also improves the patient compliance and helps in attaining such commendable eradication rates.

CONSENT FORM

Yourself Mr/Mrs/Ms_____

are being asked to be a participant in the research study titled " Anti Helicobacter Pylori therapy response in Erosive and Non erosive Gastritis – A Prospective Study in CMC Hospital, Coimbatore Medical College. You satisfy eligibility as per the inclusion criteria. You can ask any questions you may have before agreeing to participate.

Research being done

Anti Helicobacter Pylori therapy response in Erosive and Non erosive Gastritis – A Prospective Study

Purpose of Research

To identify the relationship of Helicobacter pylori with erosive and non erosive gastritis ,to study the effects of Anti H. pylori therapy and their effects on both types of gastritis.

Procedures involved

Adult patients attending the outpatient department of Coimbatore Medical College hospital with complaints of dyspepsia for more than 3 months are selected. Detailed history including personal history will be taken. Thorough clinical examination is done.

After getting informed consent ,they are subjected to upper GI endoscopy using video endoscopy-Olympus CV 150 series

After making Endoscopic diagnosis,3 biopsy samples are taken .2 samples from gastric antrum & the 3rd sample from lesion if any.

First sample is subjected to rapid urease test Second sample is stained using Giemsa special stain for Helicobacter pylori demonstration

The third sample is graded pathologically & the patient is treated with Amoxycillin ,Clarithromycin and PPI for 2 weeks followed by PPI for 4 weeks.

Patient is regularly followed up in each visit and 4 weeks after completion of treatment patient's history regarding dyspeptic symptoms noted and also endoscopic biopsy done and Rapid urease test repeated to detect H.pylori ,also the nature of gastritis reassessed whether resolved or not.

Decline from Participation

You have the option to decline from participation in the study

Privacy and Confidentiality

Privacy of individuals will be respected and any information about you or provided by you during the study will be kept strictly confidential.

Authorization to publish Results

Results of the study may be published for scientific purposes and/or presented to scientific groups, however you will not be identified.

Statement of Consent

I volunteer and consent to participate in this study.Ihave read the consent or it has been read to me.The study has been fully explained to me,and I may ask questions at any time.

Signature/left thumb impression

Date

(volunteer)

Signature of witness

Date

ஒப்புதல் படிவம்

பெயர் :

பாலினம் :

முகவரி : வயது :

அரசு கோவை மருத்துவக் கல்லூரியில் பொது மருத்துவம் மருத்துவ துறையில் பட்ட மேற்படிப்பு பயிலும் மாணவர் _____
அவர்கள்_____
ஆய்வில் மேற்கோள்ளும் செய்முறை மற்றும் அனைத்து விவரங்களையும் கேட்டுக் கொண்டு எனது சந்தேகங்களை தெளிவுபடுத்திக் கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன்.

நான் இந்த ஆய்வில் முழு சம்மதத்துடன், சுய சிந்தனையுடனும் கலந்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் என்னுடைய அனைத்து விபரங்கள் பாதுகாக்கப்படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் ஆட்சேபனை இல்லை என்பதை தெரிவித்துக்கொள்கிறேன். எந்த நேரத்தில் அந்த ஆய்விலிருந்து நான் விலகிக் கொள்ள எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

இடம் : கையொப்பம் / ரேகை

நாள் :

ABBREVIATIONS FOR MASTER CHART

SES - Socioeconomic status

CF – Clinical features

EF- Endoscopic findings

RUT- Rapid urease test

Sex : M- Male F- Female

Socioeconomic status

L- Lower class

LM – Lower Middle class

UM- Upper Middle class

U- Upper class

Clinical features

A- Abdominal pain

B- Bloating

E- Early satiety

N – Nausea

R – Anorexia

V – Vomiting

AB – Abdominal pain & Bloating

AE – Abdominal pain & early satiety

AN – Abdominal pain & nausea

AR- Abdominal pain & Anorexia

ALL – All features

Site of lesion

1- Pylorus
2- Antrum’
3- Lesser curvature
4- Diffuse

RUT & follow up RUT

1. Postive 2. Negative

MASTER CHART

sl no	age	Sex	ses	CF	EF	site	RUT	Follow up	2nd RUT	Abd pain	Bloating	Early satiety	Nausea	Anorexia	Vomiting	Erosive
1	37	M	L	AB	E	1	1	1	2	1	1					3
2	44	M	LM	N	N	1	1									
3	42	M	L	AE	E	2	1	1	2	1		1				3
4	38	M	LM	B	E	1	1	1	2		1					3
5	39	F	L	V	N	2	2								1	
6	37	M	UM	AB	E	1	1	1	2	2	1					3
7	43	F	LM	B	E	1	1									
8	42	M	L	ALL	E	2	1	1	2	1	1	1	1	1	1	3
9	40	M	LM	AB	E	1	1									
10	41	M	L	N	N	1	1	1	2				2			3
11	36	F	LM	AB	E	1	1	1	2	1	1					3
12	38	F	L	ALL	E	1	1	1	1	1	2	2	1	1	1	1
13	40	M	LM	B	E	1	2									
14	44	M	L	ALL	E	1	1								1	
15	41	M	LM	AB	E	3	1									
16	37	F	L	AE	E	1	1	1	2	2		1				3
17	39	M	LM	AE	N	1	1	1	2	1		2				3
18	40	M	L	V	E	3	1	1	2						1	3
19	36	M	L	N	E	1	1									
20	42	M	L	A	N	1	2									
21	43	M	L	AB	E	2	1	1	1	1	1					1

22	44	M	L	N	E	2	1									
23	37	M	L	A	N	3	1	1	1	2						2
24	41	M	LM	AB	N	1	1									
25	40	M	L	AE	E	1	1	1	2	1		1				3
26	42	M	L	N	E	1	1	1	2				1			3
27	43	M	L	A	E	2	1	1	2	2						3
28	44	M	L	AN	N	1	1									
29	40	F	LM	A	E	1	1									
30	39	F	UM	AB	N	2	1									
31	41	M	L	AN	E	1	1	1	1	1			1			1
32	39	F	L	B	E	1	1	1	2		1					3
33	36	M	LM	A	E	1	1									
34	42	M	LM	ALL	E	1	1	1	2	2	2	1	1	1	1	3
35	51	M	LM	AN	N	1	1									
36	50	F	L	V	E	1	1								1	
37	54	M	LM	N	N	1	2									
38	52	M	LM	AN	E	1	1	1	2	1			2			3
39	53	M	L	AE	E	1	1	1	2	1		1				3
40	46	M	UM	AB	E	1	1	1	1	2	1					1
41	49	M	LM	AN	N	1	1	1	2	1			1			3
42	54	M	UM	N	E	1	1									
43	47	F	L	AB	E	1	1	1	2	1	1					3
44	53	M	LM	AE	N	3	1	1	1	2		2				2
45	48	M	L	B	E	2	1	1	2		2					3
46	49	F	LM	AB	E	1	1	1	2	1	1					3
47	50	F	LM	ALL	E	1	1	1	1	2	1	1	1	1	1	1
48	46	M	UM	AN	N	1	2									

49	53	M	L	B	E	1	1	1	2		1					3
50	50	F	UM	AB	E	1	1	1	2	1	2					3
51	50	M	LM	AB	E	1	1	1	2	1	1					3
52	52	F	L	B	E	1	1									
53	51	F	L	B	N	1	1	1	2		1					3
54	54	M	LM	AB	E	1	1	1	2	2	2					3
55	49	F	LM	B	N	1	1	1	2		2					3
56	50	M	L	AB	E	1	1									
57	51	M	L	B	N	3	2									
58	56	F	L	ALL	E	1	1								1	
59	59	M	L	A	E	1	1	1	2	1						3
60	60	M	U	AB	E	1	1	1	2	1	1					3
61	64	F	L	AB	E	1	1									
62	65	M	UM	B	E	1	2									
63	60	M	L	AE	E	1	1	1	2	2		2				3
64	60	M	LM	AB	N	1	1	1	2	1	1					3
65	62	M	L	B	E	1	1									
66	59	F	L	V	E	1	1	1	2						1	3
67	56	M	LM	R	N	1	1	1	2							3
68	57	M	L	AE	E	1	1									
69	17	F	L	A	E	2	2									
70	22	M	L	AN	E	1	2									
71	24	F	LM	AB	E	1	1	1	2	1	2					3
72	19	M	L	AN	N	1	1	1	2	1						3
73	20	M	L	AB	E	1	1									
74	28	F	L	AB	E	2	1	1	2	1	1					3
75	32	M	L	AN	E	1	1									

76	33	M	LM	AB	E	2	1	1	2	1	2					3
77	34	F	LM	AE	E	1	1	1	2	2		1				3
78	34	M	LM	V	N	3	1	1	2							3
79	30	M	L	AR	E	2	1									
80	33	M	LM	A	E	1	1	1	2	1						3
81	32	M	L	AB	E	1	1	1	2	1	1					3